U.S. DEPARTMENT OF COMMERCE
National Technical Information Service

PB-292 426

Ambient Water Quality Criteria Chlorinated Naphthalenes

(U.S.) Environmental Protection Agency, Washington, DC

1978

PB 292426

CHLORINATED NAPHTHALENES

Ambient Water Quality Criteria

Criteria and Standards Division
Office of Water Planning and Standards
U.S. Environmental Protection Agency
Washington, D.C.

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

CRITERION DOCUMENT

CHLORINATED NAPHTHALENES

Criteria

Aquatic Life

For 1-chloronaphthalene, the criterion to protect freshwater aquatic life, as derived using procedures other than the Guidelines, is 29 µg/l as a 24-hour average and the concentration should never exceed 67 µg/l at any time.

For 1-chloronaphthalene, the criterion to protect saltwater aquatic life, as derived using the Guidelines, is 2.8 µg/l as a 24-hour average and the concentration should never exceed 6.4 µg/l at any time.

Human Health

For the protection of human health from the toxic properties of chlorinated naphthalenes ingested through water and through contaminated aquatic organisms, the ambient water criteria for the various classes of chlorinated naphthalenes are:

	Criterion Level (µg/l)
Trichloronaphthalenes	3.9
Tetrachloronaphthalenes	1.5
Pentachloronaphthalenes	0.39
Hexachloronaphthalenes	0.15
Octachloronaphthalene	0.08

BIBLIOGRAPHIC INFORMATION

PB-292 426

Ambient Water Quality Criteria: Chlorinated Naphthalenes.

1978

PERFORMER: Environmental Protection Agency, Washington, DC. Criteria and Standards Div.

Section 304(a) of the Clean Water Act (33 U.S.C. 1314(a)), requires EPA to publish and periodically update water quality criteria. These criteria are to reflect the latest scientific knowledge on the identifiable effects of pollutants on public health and welfare, aquatic life, and recreation. This report presents water quality criteria for chlorinated naphthalenes. It presents concentration criteria for the protection of fresh water and saltwater aquatic life. It presents 'safe' concentrations for humans, and in the case of suspect or proven carcinogens, gives various levels of incremental cancer risk. A section 304(a) water quality criterion is a qualitative or quantitative estimate of the concentration of a water constituent or pollutant in ambient waters which, when not exceeded, will ensure a water quality sufficient to protect a specified water use. Under the Act a criterion is a scientific entity, based solely on data and scientific judgment. It does not reflect considerations of economic or technological feasibil, ...

KEYWORDS: *Water quality, *Toxicology, *Water pollution effects(Humans), *Water pollution effects(Animals), *Toxic substances, *Naphthalene/chloro.

Available from the National Technical Information Service, Springfield, Va. 22161

PRICE CODE: PC A04/MF A01

Introduction

Chlorinated naphthalenes consist of two fused six carbon-membered aromatic rings where any or all of the eight hydrogen atoms can be replaced with chlorine. Theoretically, 76 individual isomers are possible and may exist. The commercial products are usually mixtures with various degrees of chlorination, and are presently manufactured and marketed in the United States under the trade name, Halowaxes^{R}.

Mixtures of tri- and tetrachloronaphthalenes (solids) comprise the bulk of market use as the paper impregnant in automobile capacitors. Less use is made of mixtures of the mono- and dichloronaphthalenes as oil additivies for engine cleaning, and in fabric dyeing. In 1956, the total United States production of chlorinated naphthalenes was approximately 3,175 metric tons (Hardie, 1964).

Possible impurities of these products are chlorinated derivatives, corresponding to the impurities in coal tar, or petroleum-derived naphthalene feedstocks which may include biphenyls, fluorenes, pyrenes, anthracenes, and dibenzofurans.

The potential for environmental exposure may be significant when these compounds are used as oil additives, in the electroplating industry, and in the fabric dyeing industry. The extent of leaching of chlorinated naphthalenes from discarded capacitors and old cable insulation (manufactured prior to curtailment of the chemical's use in such products) has not been determined.

Chlorinated naphthalenes have been detected as a contaminant in foreign commercial PCB formulations (Phenoclor,

Clophen, and Kanechlor) along with chlorinated dibenzofurans, and are present in domestic PCBs (Aroclors) but at lower levels than in foreign formulations (Vos, et al. 1970; Bowes, et al. 1975; Roach and Pomerantz, 1974).

The synthesis of chlorinated naphthalenes generally involves the chlorination of naphthalene by chlorine in the presence of catalytic amounts of ferric or antimony chloride. This production process yields mixtures of highly chlorinated naphthalenes in varying quantities by further chlorination of the lesser substituted products. Only 1-chloronaphthalene and octachloronaphthalene are readily isolated from the products of direct chlorination (Hardie, 1964). All of the possible two monochloro-, 10 dichloro-, and 14 trichloronaphthalenes have been isolated and identified. However, not all of the tetra- and higher chloro-isomers have been characterized.

Table 1 presents physical property data for all of the chlorinated naphthalenes which have been isolated and identified. The physical properties of the chlorinated naphthalenes are generally dependent on the degree of chlorination. Melting points of the pure compounds range from 17 degrees C for 1-chloronaphthalene to 198 degrees C for 1,2,3,4-tetrachloronaphthalene (Hardie, 1964). Also, as the degree of chlorination increases, the specific gravity, boiling point, fire and flash points all increase, while the vapor pressure and water solubility decrease (Hardie, 1964). Mixtures of the mono- and dichloronaphthalenes are generally liquid at room temperature, whereas mixtures of

the more highly chlorinated naphthalenes tend to be waxy solids (Howard and Durkin, 1973).

Chlorinated naphthalenes, like PCBs, exhibit a high degree of chemical and thermal stability as indicated by their resistance to most acids and alkalies and to dehydrochlorination (Kover, 1975).

Limited data exist on the toxicity of chlorinated naphthalenes toward aquatic organisms. Only two pure isomers, 1-chloronaphthalene and 1,2,3,4,5,6,7,8-octachloronaphthalene, have been tested in freshwater aquatic organisms. Results from bioassays on these compounds show that the monochloro-isomer is more acutely toxic than the octachloro-isomer for a freshwater plant, a freshwater invertebrate species, and a freshwater vertebrate species.

The same trend in acute toxicity for the mono- and octachloro-isomers exists in saltwater organisms. An embryo-larval chronic toxicity test conducted on a saltwater vertebrate species for 1-chloronaphthalene demonstrated chronic toxic effects. No other chronic data exist for any other chlorinated naphthalene for any other freshwater or saltwater species.

A considerable amount of acute toxicity data on chlorinated naphthalene mixtures (Halowaxes) for saltwater organisms has been compiled. Reported acute 96 hr. LC50 values for invertebrate species do not suggest a trend in toxicity versus degree of mixture chlorination. Other toxicity data for saltwater organisms also do not suggest a consistent trend in toxicity with an increased degree of mixture chlorination.

TABLE 1

Physical properties of chloronaphthalenes (Hardie, 1964)

Isomer	Mp degrees C	Bp degrees C	density$^{\text{temp.}(^{\circ}C)}$
1-chloronaphthalene	Ca.17	259.3	1.1938^{20}
2-chloronaphthalene	61	265	1.2656^{16}
1,2-dichloronaphthalene	35		$1.3147^{48.5}$
1,3-dichloronaphthalene	61.5	291 (755 mm Hg)	
1,4-dichloronaphthalene	67.5	287	$1.2997^{75.9}$
1,5-dichloronaphthalene	106.5		
1,6-dichloronaphthalene	48.5		
1,7-dichloronaphthalene	63.5	285.5	$1.2611^{99.5}$
1,8-dichloronaphthalene	88.5		$1.2924^{99.8}$
2,3-dichloronaphthalene	135	285	
2,6-dichloronaphthalene	120		
2,7-dichloronaphthalene	114		
1,2,3-trichloronaphthalene	81		
1,2,4-trichloronaphthalene	92		
1,2,5-trichloronaphthalene	78		
1,2,6-trichloronaphthalene	92.5		
1,2,7-trichloronaphthalene	88		
1,2,8-trichloronaphthalene	83		
1,3,5-trichloronaphthalene	94		
1,3,6-trichloronaphthalene	80.5		
1,3,7-trichloronaphthalene	113		

1,3,8-trichloronaphthalene	89.5
1,4,5-trichloronaphthalene	133
1,4,6-trichloronaphthalene	65
2,3,5-trichloronaphthalene	109.5
2,3,6-trichloronaphthalene	90.5
1,2,3,4-tetrachloronaphthalene	198
1,3,5,8-tetrachloronaphthalene	131
1,4,6,7-tetrachloronaphthalene	139
1,2,3,4,5-pentachloronaphthalene	168.5
1,2,3,4,5,6,8-heptachloronaphthalene	194
1,2,3,4,5,6,7,8-octachloronaphthalene	192

Halowaxes bioconcentrate in saltwater algal and saltwater invertebrate species 25 to 2,300 fold with no consistent trend for the magntude of the bioconcentration with respect to degree of mixture chlorination (Walsh, et al. 1977; U.S. EPA, 1976).

Chlorinated naphthalenes demonstrate acute and chronic toxic effects for a large variety of non-human mammals including rats (Bennett, et al. 1938), rabbits (Hambrick, 1957), pigs (Link, et al. 1958), cattle (Olson, 1969), and sheep (Brock, et al. 1957). Generally the mono- and dichloro-isomers are only slightly toxic, the tri-, tetra-, penta- and hexa-isomers are the most toxic and the octachloro-isomer generally the least toxic in these studies. Prevalent pathological symptoms include hyperkeratosis and damage to the liver and kidney of each species. Toxicity is caused either by ingestion, inhalation, or dermal application of the toxicant.

A similar situation for chlorinated naphthalene toxicity in humans has been demonstrated (Hambrick, 1957; McLetchie and Robertson, 1942; Kleinfeld, et al. 1972; Cotter, 1944; Greenburg, et al. 1939). Toxicity can be caused by dermal contact, inhalation, and presumably ingestion. The prevalent pathological symptoms are liver injury, changes in serum enzyme levels, and dermal manifestations such as chloracne. The primary hepatotoxic isomers for man seem to be penta- and hexachloronaphthalene (Am. Ind. Hyg. Assoc. 1966). The higher chlorinated naphthalenes appear to be the most toxic for dermal exposure.

In several mammalian species, the chlorinated naphthalenes are metabolized to some extent to chlorinated naphthols, and to some extent are excreted unchanged (Cornish and Block, 1958; Ruzo, et al. 1976a,b). These studies indicate that as the degree of isomer chlorination increases, the extent of isomer metabolism to chlorinated naphthols decreases with no metabolism of pentachloro- and higher chlorinated isomers apparent (Cornish and Block, 1958; Ruzo, et al. 1976a). Howard and Durkin (1973) report that the available data on metabolism coupled with the chemical and physical similarities to polychlorinated biphenyls indicate that the higher chlorinated naphthalenes are relatively stable and are likely to persist when released to the environment.

REFERENCES

American Industrial Hygiene Association. 1966. Chloronaphthalenes. Hyg. Guide Ser. Jan-Feb.

Bennett, G.A., et al. 1938. Morphological changes in the liver of rats resulting from exposure to certain chlorinated hydrocarbons. Jour. Ind. Hyg. Toxicol. 20: 97.

Bowes, G.W., et al. 1975. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 256: 305.

Brock, W.E., et al. 1957. Chlorinated naphthalene intoxication in sheep. Am. Jour. Vet. Res. 18: 625.

Cornish, H.H., and W.D. Block. 1958. Metabolism of chlorinated naphthalenes. Jour. Biol. Chem. 231: 583.

Cotter, L.H. 1944. Pentachlorinated naphthalenes in industry. Jour. Am. Med. Assoc. 125: 373.

Greenburg, L., et al. 1939. The systemic effects resulting from exposures to certain chlorinated hydrocarbons. Jour. Ind. Hyg. Toxicol. 21: 29.

Hambrick, G.W. 1957. The effect of substituted naphthalenes on the pilosebaceous apparatus of rabbit and man. Jour. Invest. Dermat. 28: 89.

Hardie, D.W.F. 1964. Chlorocarbons and chlorohydrocarbons: chlorinated naphthalenes. Pages 297-303 in Kirk-Othmer encyclopedia of chemical technology. 2nd ed. John Wiley and Sons. Inc., New York.

Howard, P.H., and P.R. Durkin. 1973. Preliminary environmental hazard assessment of chlorinated napthalenes, silicones, fluorocarbons, benzenepolycarboxylates, and chlorophenols. EPA Publ. No. 560/2-74-001. U.S. Environ. Prot. Agency. Washington, D.C.

Kleinfeld, M., et al. 1972. Clinical effects of chlorinated naphthalene exposure. Jour. Occup. Med. 14: 377.

Kover, F.D. 1975. Environmental hazard assessment report: chlorinated naphthalenes. EPA Publ. No. 560/8-75-001. U.S. Environ. Prot. Agency. Washington, D.C.

Link, R.R., et al. 1958. Toxic effect of chlorinated naphthalenes in pigs. Jour. Am. Vet. Med. Assoc. 133: 83.

McLetchie, N.G.R., and D. Robertson. 1942. Chlorinated naphthalene poisoning. Br. Med. Jour. 1: 691.

Olson, C. 1969. Bovine hyperkeratosis (X-disease, highly chlorinated naphthalene poisoning). Historical review in C.A. Bradley and C.E. Cornelius, eds. Advances in veterinary science and comparative medicine. Academic Press, New York.

Roach, J.A.G. and I.H. Pomerantz. 1974. The finding of chlorinated dibenzofurans in a Japanese polychlorinated biphenyl sample. Bull. Environ. Contam. Toxicol. 12: 338.

Ruzo, L., et al. 1976a. Metabolism of chlorinated naphthalenes. Jour. Agric. Food Chem. 24: 581.

Ruzo, L., et al. 1976b. Uptake and distribution of chloronaphthalenes and their metabolites in pigs. Bull. Environ. Contam. Toxicol. 16: 233.

U.S. EPA. 1976. Semi-annual report, Environ. Res. Lab., Gulf Breeze, Fla. April-September, 1976. U.S. Environ. Prot. Agency.

Vos, J.G., et al. 1970. Identification and toxicological evaluation of chlorinated dibenzofurans and chlorinated naphthalenes in two commercial polychlorinated biphenyls. Food Cosmet. Toxicol. 8: 625.

Walsh, G.E., et al. 1977. Effects and uptake of chlorinated naphthalenes in marine unicellular algae. Bull. Environ. Contam. Toxicol. 18: 297.

AQUATIC LIFE TOXICOLOGY*

FRESHWATER ORGANISMS

Introduction

The only chlorinated naphthalenes for which data are available for freshwater organisms are 1-chloronaphthalene and octachloronaphthalene. The available LC50 and EC50 values for the bluegill, Daphnia magna, and an alga indicate similar sensitivity of these groups.

Acute Toxicity

The 96-hour LC50 for the bluegill and 1-chloronaphthalene is 2,270 µg/l (Table 1). After adjustment for testing methodology and species sensitivity according to the Guidelines, the Final Fish Acute Value is 320 µg/l. A single test with Daphnia magna and the same chemical (U.S. EPA, 1978) provides a 48-hour EC50 of 1,600 µg/l (Table 2) and a Final Invertebrate Acute Value of 67 µg/l. The latter becomes the Final Acute Value.

Chronic Toxicity

No embryo-larval or life-cycle tests have been conducted with freshwater fish or invertebrate species and any chlorinated naphthalene.

*The reader is referred to the Guidelines for Deriving Water Quality Criteria for the Protection of Aquatic Life (43 FR 21506 (May 18, 1978) and 43 FR 29028 (July 5, 1978)) and the Methodology Document in order to better understand the following discussion and recommendation. The following tables contain the appropriate data that were found in the literature, and at the bottom of each table are the calculations for deriving various measures of toxicity as described in the Guidelines.

Plant Effects

The alga, Selenastrum capricornutum, has been exposed to 1-chloronaphthalene and the 96-hour EC50 values for chlorophyll a and cell numbers are 1,030 and 1,000 µg/l, respectively (Table 3).

Miscellaneous

A variety of acute tests of the effects of octachloronaphthalene have been conducted with the bluegill, Daphnia magna, and an alga (U.S. EPA, 1978). No adverse effects were observed at concentrations as high as 500,000 to 600,000 µg/l (Table 4).

CRITERION FORMULATION

Freshwater - Aquatic Life

Summary of Available Data

The concentrations below have been rounded to two significant figures.

1-chloronaphthalene

Final Fish Acute Value = 320 µg/l

Final Invertebrate Acute Value = 67 µg/l

Final Acute Value = 67 µg/l

Final Fish Chronic Value = not available

Final Invertebrate Chronic Value = not available

Final Plant Value = 1,000 µg/l

Residue Limited Toxicant Concentration = not available

Final Chronic Value = 1,000 µg/l

0.44 x Final Acute Value = 29 µg/l

No freshwater criterion can be derived for any chlorinated naphthalene using the Guidelines because no Final Chronic Value for either fish or invertebrate species or a good substitute for either value is available.

Data for 1-chloronaphthalene and saltwater organisms can be used to estimate a criterion.

For 1-chloronaphthalene and saltwater organisms, 0.44 times the Final Acute Value is less than the Final Chronic Value derived from results of an embryo-larval test with the sheepshead minnow. Therefore, a reasonable estimate of a criterion for 1-chloronaphthalene and freshwater organisms would be 0.44 times the Final Acute Value.

The maximum concentration for 1-chloronaphthalene is the Final Acute Value of 67 µg/l and the 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on freshwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

CRITERION: For 1-chloronaphthalene the criterion to protect freshwater aquatic life as derived using procedures other than the Guidelines is 29 µg/l as a 24-hour average and the concentration should not exceed 67 µg/l at any time.

Table 1. Freshwater fish acute values for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Chemical Description	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Bluegill, *Lepomis macrochirus*	S	U	1-chloro-naphthalene	96	2,270	1,240

* S = static

** U = unmeasured

Geometric mean of adjusted values: 1-chloronaphthalene = 1,240 µg/l $\frac{1,240}{3.9}$ = 320 µg/l

Table 2. Freshwater invertebrate acute values for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Chemical Description	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Cladoceran, Daphnia magna	S	U	1-chloro-naphthalene	48	1,600	1,400

* S = static

** U = unmeasured

Geometric mean of adjusted values: 1-chloronaphthalene = 1,400.µg/l $\frac{1,400}{21}$ = 67 µg/l

Table 3. Freshwater plant effects for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Effect	Concentration (ug/l)
	1-chloronaphthalene	
Alga, Selenastrum capricornutum	EC50 96-hr chloropyll a	1,030
Alga, Selenastrum capricornutum	EC50 96-hr cell numbers	1,000

Lowest plant value: 1-chloronaphthalene = 1,000 ug/l

Table 4. Other freshwater data for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Test Duration	Effect	Result µg/l
		Octachloronaphthalene	
Alga, *Selenastrum capricornutum*	96 hrs	EC50 chlorophyll *a*	>500,000
Alga, *Selenastrum capricornutum*	96 hrs	EC50 cell numbers	>500,000
Cladoceran, *Daphnia magna*	48 hrs	LC50	>530,000
Bluegill, *Lepomis macrochirus*	96 hrs	LC50	>600,000

SALTWATER ORGANISMS

Introduction

Most of the data concerning the effects of chlorinated naphthalenes on saltwater organisms are for commercial mixtures of mono- through hexachloronaphthalenes in different proportions. These results do not appear to be useful in the derivation of criteria for specific chlorinated naphthalenes, and the data for these mixtures are included in the tables for information purposes. Most of the remaining data are for 1-chloronaphthalene. These results are very similar to those freshwater data for a fish, an invertebrate, and an alga using comparable test procedures (U.S. EPA, 1978).

Acute Toxicity

The sheepshead minnow has been exposed to 1-chloronaphthalene (U.S. EPA, 1978) and the 96-hour LC50, after adjustment using the appropriate Guidelines factors, is 1,290 µg/l (Table 5). Adjustment for species sensitivity results in a Final Fish Acute Value of 350 µg/l. Of the saltwater invertebrate species, only the mysid shrimp has been tested with 1-chloronaphthalene. The adjusted 96-hour LC50 is 313 µg/l (Table 6), which indicates a greater sensitivity than the sheepshead minnow. After this result is divided by the sensitivity factor of 49, the Final Invertebrate Acute Value is 6.4 µg/l. This concentration also becomes the Final Acute Value since this invertebrate species value is lower than the equivalent value for fish.

Chronic Toxicity

An embryo-larval test has been conducted with the sheepshead minnow and 1-chloronaphthalene (U.S. EPA, 1978). The chronic value is 329 µg/l and this results in a Final Fish Chronic Value of 49 µg/l after division by the sensitivity factor (Table 7). No other chronic data are available so 49 µg/l also becomes the Final Chronic Value for 1-chloronaphthalene.

Plant Effects

The 96-hour EC50 values for chlorophyll a and cell numbers of the alga, Skeletonema costatum are 1,130 and 1,300 µg/l, respectively for 1-chloronaphthalene (Table 8).

Residues

The only available equilibrium residue datum (Table 9) for chlorinated naphthalenes is for Halowax 1014, a mixture of tetra-, penta-, and hexachloronaphthalene (U.S. EPA, 1976). The bioconcentration factor for this mixture is 2,300 which indicates a need for comparable data on individual chlorinated naphthalenes.

Miscellaneous

As with the freshwater species, the acute toxicity results for the sheepshead minnow, mysid shrimp, and an alga (U.S. EPA, 1978) were all greater than 500,000 µg/l for octachloronaphthalene (Table 10). A great variety of other data is available for various mixtures of chlorinated naphthalenes and bioconcentration, inhibition of algal growth, intermolt time for crabs, and other effects (Table 10).

CRITERION FORMULATION

Saltwater - Aquatic Life

Summary of Available Data

The concentrations below have been rounded to two significant figures.

1-chloronaphthalene

Final Fish Acute Value = 350 µg/l

Final Invertebrate Acute Value = 6.4 µg/l

Final Acute Value = 6.4 µg/l

Final Fish Chronic Value = 49 µg/l

Final Invertebrate Chronic Value = not available

Final Plant Value = 1,100 µg/l

Residue Limited Toxicant Concentration = not available

Final Chronic Value = 49 µg/l

0.44 x Final Acute Value = 2.8 µg/l

The commercial mixtures of chlorinated naphthalenes are not considered in the development of a criterion since the toxicity of each chlorinated naphthalene in the mixtures may be different, and different proportions of these individual chemicals would have different toxicity.

The maximum concentration of 1-chloronaphthalene is the Final Acute Value of 6.4 µg/l and the 24-hour average concentration is 0.44 times the Final Acute Value. No important adverse effects on saltwater aquatic organisms have been reported to be caused by concentrations lower than the 24-hour average concentration.

CRITERION: For 1-chloronaphthalene the criterion to protect saltwater aquatic life as derived using the Guidelines is 2.8 µg/l as a 24-hour average and the concentration should not exceed 6.4 µg/l at any time.

Table 5. Marine fish acute values for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Bioassay Method*	Test Conc.**	Chemical Description	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)
Sheepshead minnow, Cyprinodon variegatus	S	U	1-chloro-naphthalene	96	2,360	1,290

* S = static

** U = unmeasured

Geometric mean of adjusted value = 1,290 µg/l $\frac{1,290}{3.7}$ = 350 µg/l

Table 6. Marine invertebrate acute values for chlorinated naphthalenes

Organism	Bioassay Method*	Test Conc.**	Chemical Description	Time (hrs)	LC50 (ug/l)	Adjusted LC50 (ug/l)	Reference
Mysid shrimp, Mysidopsis bahia	S	U	1-chloro-naphthalene	96	370	313	U.S. EPA, 1978
Brown shrimp, Penaeus aztecus	FT	M	Halowax 1014***	96	7.5	7.5	U.S. EPA, 1976
Grass shrimp, Palaemonetes pugio	FT	M	Halowax 1014***	96	248	248	U.S. EPA, 1976
Grass shrimp (post-larva), Palaemonetes pugio	R	M	Halowax 1000****	96	440	484	Green & Neff, 1977
Grass shrimp (adult), Palaemonetes pugio	R	M	Halowax 1000****	96	325	358	Green & Neff, 1977
Grass shrimp (post-larva), Palaemonetes pugio	R	M	Halowax 1013*****	96	74	81	Green & Neff, 1977
Grass shrimp (post-larva), Palaemonetes pugio	R	M	Halowax 1099******	96	69	76	Green & Neff, 1977
Grass shrimp (adult), Palaemonetes pugio	R	M	Halowax 1099******	96	90	99	Green & Neff, 1977

* S = static; FT = flow-through; R = renewal

** M = measured; U = unmeasured

*** Halowax® 1014: 20% tetrachloronaphthalene, 40% pentachloronaphthalene, 40% hexachloronaphthalene

**** Halowax® 1000: 60% monochloronaphthalene, 40% dichloronaphthalene

***** Halowax® 1013: 10% trichloronaphthalene, 50% tetrachloronaphthalene, 40% pentachloronaphthalene

****** Halowax® 1099: 10% dichloronaphthalene, 40% trichloronaphthalene, 40% tetrachloronaphthalene, 10% pentachloronaphthalene

Geometric mean of adjusted values for 1-chloronaphthalene = 313 µg/l $\frac{313}{49}$ = 6.4 µg/l

Table 7. Marine fish chronic values for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Test*	Limits (ug/l)	Chronic Value (ug/l)
		1-chloronaphthalene	
Sheepshead minnow, Cyprinodon variegatus	E-L	460-940	329

*E-L = embryo-larval

Geometric mean of chronic value = 329 µg/l $\frac{329}{6.7}$ = 49 µg/l 1-chloronaphthalene

Lowest chronic value = 329 µg/l

Table 8. Marine plant effects for chlorinated naphthalenes (U.S. EPA, 1978)

Organism	Effect	Concentration (ug/l)
		1-chloronaphthalene
Alga, *Skeletonema costatum*	Chlorophyll a EC50 after 96 hr	1,130
Alga, *Skeletonema costatum*	Cell numbers EC50 after 96 hr	1,300

Lowest plant value = 1,130 ug/l

Table 9. Marine residues for chlorinated naphthalenes (U.S. EPA, 1976)

Organism	Bioconcentration Factor	Time (days)
	Halowax 1014*	
Brown shrimp, *Penaeus aztecus*	2,300	4

*Halowax 1014: 20% tetrachloronaphthalene, 40% pentachloronaphthalene, 40% hexachloronpahthalene

Table 10. Other marine data for chlorinated naphthalenes

Organism	Test Duration	Effect	Result µg/l	References
Alga, *Chlorococcum* sp.	24 hrs	Bioconcentration factor = 25-32*	-	Walsh, et al. 1977
Alga, *Chlorococcum* sp.	24 hrs	Bioconcentration factor = 60-120**	-	Walsh, et al. 1977
Alga, *Chlorococcum* sp.	24 hrs	Bioconcentration factor = 110-140***	-	Walsh, et al. 1977
Alga, *Chlorococcum* sp.	7 days	11.7% inhibition of growth*	500	Walsh, et al. 1977
Alga, *Chlorococcum* sp.	7 days	45.8% inhibition of growth*	1,000	Walsh, et al. 1977
Alga, *Dunaliella tertiolecta*	7 days	11% inhibition of growth*	100	Walsh, et al. 1977
Alga, *Dunaliella tertiolecta*	7 days	18.6% inhibition of growth*	500	Walsh, et al. 1977
Alga, *Dunaliella tertiolecta*	7 days	43% inhibition of growth*	1,000	Walsh, et al. 1977
Alga, *Nitzschia* sp.	7 days	17.1% inhibition of growth*	500	Walsh, et al. 1977
Alga, *Nitzschia* sp.	7 days	42.3% inhibition of growth*	1,000	Walsh, et al. 1977
Alga, *Nitzschia* sp.	7 days	13.2% inhibition of growth**	500	Walsh, et al. 1977
Alga, *Nitzschia* sp.	7 days	16.6% inhibition of growth**	1,000	Walsh, et al. 1977
Alga, *Skeletonema costatum*	96 hrs	Chlorophyll a EC50*****	>500,000	U.S. EPA, 1978
Alga, *Skeletonema costatum*	96 hrs	Cell numbers EC50*****	>500,000	U.S. EPA, 1978
Alga, *Thalassiosira pseudonana*	7 days	21.3% inhibition of growth*	500	Walsh, et al. 1977

Table 10. (Continued)

Organism	Test Duration	Effect	Result µg/l	References
Alga, *Thalassiosira pseudonana*	7 days	48.4% inhibition of growth*	1,000	Walsh, et al. 1977
Alga, *Thalassiosira pseudonana*	7 days	7.1% inhibition of growth**	1,000	Walsh, et al. 1977
Horseshoe crab, *Limulus polyphemus*	27 days	Time required for 50% mortality (LT50) of T_1 stage larvae****	80	Neff & Giam, 1977
Horseshoe crab, *Limulus polyphemus*	-	Average length of time of intermolt between T_2 and T_3 stages reduced by 3.4 days****	40	Neff & Giam, 1977
Horseshoe crab, *Limulus polyphemus*	-	Average length of time of intermolt between T_3 and T_4 stages reduced by 14.8 days****	20	Neff & Giam, 1977
Horseshoe crab, *Limulus polyphemus*	-	Average length of time of intermolt between T_3 and T_4 stages reduced by 16.8 days****	20	Neff & Giam, 1977
Horseshoe crab, *Limulus polyphemus*	-	Average length of time of intermolt between T_3 and T_4 stages reduced by 18.4 days****	80	Neff & Giam, 1977
Horseshoe crab, *Limulus polyphemus*	-	Increased rates of respiration of T_1 and T_2 stages****	20 and 40	Neff & Giam, 1977
Grass shrimp, *Palaemonetes pugio*	15 days	Bioconcentration factor = 63*	-	Green & Neff, 1977
Grass shrimp, *Palaemonetes pugio*	12 days	Bioconcentration factor = 187**	-	Green & Neff, 1977
Grass shrimp, *Palaemonetes pugio*	5 days	Bioconcentration factor = 257****	-	Green & Neff, 1977
Mysid shrimp, *Mysidopsis bahia*	96 hrs	LC50*****	>500,000	U.S. EPA, 1978

Table 10. (Continued)

Organism	Test Duration	Effect	Result µg/l	References
Mud crab, Rhithropanopeus harrisi	13 days	Slightly lowered survival of larvae to megalopa*	300	Neff, et al. 1977
Mud crab, Rhithropanopeus harrisi	27 days	15% survival of larvae to megalopa****	100	Neff, et al. 1977
Mud crab, Rhithropanopeus harrisi	--	Length of intermolt time from 4th zoeal molt to megalopa stage extended to 2.9 days*	300	Neff, et al. 1977
Mud crab, Rhithropanopeus harrisi	--	Length of intermolt time from 4th zoeal molt to megalopa stage extended by 4.9 days****	100	Neff, et al. 1977
Mud crab, Rhithropanopeus harrisi	--	Supernumerary zoeae (a fifth zoeal stage)****	100	Neff, et al. 1977
Mub crab, Rhithropanopeus harrisi	--	Deformed megalopa (eyestalks and appendages malformed)*	300	Neff, et al. 1977
Mud crab, Rhithropanopeus harrisi	--	Deformed megalopa (eyestalks and appendages malformed)****	100	Neff, et al. 1977
Sheepshead minnow, Cyprinodon variegatus	96 hrs	LC50***	>343	U.S. EPA, 1976
Sheepshead minnow, Cyprinodon variegatus	96 hrs	LC50*****	>560,000	U.S. EPA, 1978
Striped mullet (juvenile) Mugil cephalus	96 hrs	LC50***	>263	U.S. EPA, 1976

* Halowax 1000: 60% monochloronaphthalene, 40% dichloronaphthalene
** Halowax 1013: 10% trichloronaphthalene, 50% tetrachloronaphthalene, 40% pentachloronaphthalene
*** Halowax 1014: 20% tetrachloronaphthalene, 40% pentachloronaphthlene, 40% hexachloronaphthalene
**** Halowax 1099: 40% trichloronaphthalene, 40% tetrachloronaphthalene, 10% pentachloronaphthalene, 10% dichloronaphthalene
***** Octachloronaphthalene

REFERENCES

Green, F.A., Jr., and J.M. Neff. 1977. Toxicity, accumulation, and release of three polychlorinated naphthalenes (Halowax 1000, 1013, and 1099) in postlarval and adult grass shrimp, Palaemonetes pugio. Bull. Environ. Contam. Toxicol. 14: 399.

Neff, J.M., and C.S. Giam. 1977. Effects of Aroclor 1016 and Halowax 1099 on juvenile horseshoe crabs, Limulus polyphemus. Pages 21-36 in F.J. Vernberg, et al.,eds. Physiological responses of marine biota to pollutants. Academic Press, N.Y.

Neff, J.M., et al. 1977. Effects of polychlorinated biphenyls, polychlorinated naphthalenes and phthalate esters on larval development of the mud crab, Rhithropanopeus harrisii. Pages 95-110 in C.S. Giam, ed. Pollutant effects on marine organisms. Lexington Books, D.C. Heath and Co., Lexington, Mass.

U. S. EPA. 1976. Semi-annual report, Environ. Res. Lab., Gulf Breeze, Fla. April - September, 1976. U. S. Environ. Prot. Agency.

U. S. EPA. 1978. In-depth studies on health and environmental impacts of selected water pollutants. Contract No. 68-01-4646. U.S. Environ. Prot. Agency. Washington, D.C.

Walsh, G.E., et al. 1977. Effects and uptake of chlorinated naphthalenes in marine unicellular algae. Bull. Environ. Contam. Toxicol. 18: 297.

Mammalian Toxicology and Human Health Effects

Introduction

Polychlorinated naphthalenes have been used in various industrial processes since the turn of the century. Peak use of these compounds occurred during World War I in Germany where they were used in place of rubber, and in the United States during World War II where they were used to a large extent in heat-resistant electrical insulation. Since then many uses of polychlorinated naphthalenes have been replaced by a growing variety of plastics. In 1956 production and utilization of polycholorinated naphthalenes in the United States had decreased to approximately 3,200 metric tons per year. By 1972 production had decreased further to approximately 2,300 metric tons per year. At the present, Halochem, Inc. in Boonton, N.J., is the only manufacturer of polychlorinated naphthalenes in the United States. Amounts of chlorinated naphthalenes processed in 1978 were less than 22 metric tons for monochloronaphthalene, less than 45 metric tons total for di-, tri-, and tetrachloronaphthalene, less than 1 metric ton for pentachloronaphthalene, and virtually zero for the more highly chlorinated naphthalenes (R. Cuozzo, 1978, President, Halochem, Inc., personal communication). Projected production for 1979 totals less than 270 metric tons with 20 percent of this total expected to be monochloronaphthalene, less than 5 percent pentachloronaphthalene, and none of the more highly chlorinated naphthalenes. Although

several foreign companies manufacture polychlorinated naphthalenes, there are no known imports of these compounds. Because of their chemical and thermal stability, dielectric properties, and low viscosity in a liquid state, polychlorinated naphthalenes are still used as engine oil additives, cutting oil additives, capacitor dielectrics, and electroplating stopoff compounds. They are also used to some extent in the production of fabric dyes. In the past, polychlorinated naphthalenes have been used as pesticides, waterproofing and flame retardent compounds, and cable-covering materials.

During World Wars I and II, the industrial use of polychlorinated naphthalenes was implicated in many cases of chloracne and, to a lesser extent, liver disease. The purpose of this report is to summarize information on the occurrence, pharmacokinetic properties, and health effects of polychlorinated naphthalenes in an effort to set a criterion for acceptable levels of polychlorinated naphthalenes in water.

EXPOSURE

Polychlorinated naphthalenes do not occur naturally in the environment. Potential environmental accumulation can occur around points of manufacture of polychlorinated naphthalenes or products containing them, near sites of disposal of polychlorinated naphthalene-containing wastes, and, since polychlorinated biphenyls (PCBs) are to some extent contaminated by polychlorinated naphthalenes (Vos, et al. 1970; Bowes, et al. 1975), near sites of heavy polychlorinated biphenyl contamination. Because polychlorinated

naphthalenes are relatively insoluble in water, they would not be expected to migrate far from their point of disposition.

Currently available industrially produced polychlorinated naphthalenes occur as mixtures of various isomers as noted in Table 1 (Brinkman and Reymer, 1976). These mixtures are marketed by Koppers, Inc. under the trade name Halowax.®

TABLE 1

Approximate Compositions (WT.-%) of Halowaxes (PCN's) (Brinkman and Reymer, 1976)

Halowax	Type of PCN Mono-	Di-	Tri-	Tetra-	Penta-	Hexa-	Hepta-	Octa-
1031	95	5						
1000	60	40						
1001		10	40	40	10			
1099		10	40	40	10			
1013			10	50	40			
1014				20	40	40		
1051							10	90

Ingestion from Water and Food

To date polychlorinated naphthalenes have not been identified in either drinking water or market basket foods. Polychlorinated naphthalenes have been found in waters or sediments adjacent to point sources or areas of heavy polychlorinated biphenyl contamination (Table 2).

Polychlorinated naphthalene-contaminated sediments occur less frequently than polychlorinated biphenyl-contaminated sediments. Law and Goerlitz (1974) found polychlorinated naphthalenes in only 1 of 39 sediment samples from streams emptying into San Francisco Bay. In contrast, 97 percent of the samples contained measurable levels of polychlorinated biphenyls.

Polychlorinated naphthalenes do appear to be biomagnified in the aquatic ecosystem. As noted in Table 2, Crump-Wiesner, et al. (1973) found that concentrations of polychlorinated naphthalenes in sediments were 220- to 877-fold greater than in the water overlying these sediments. Erickson, et al. (1978), however, found a polychlorinated naphthalene level in contaminated sediments near a capacitor factory that was only six-fold greater than the level in the overlying water. Algae definitely accumulate polychlorinated naphthalenes. Walsh, et al. (1977) have found polychlorinated naphthalene levels in algae that were 24 to 140-fold higher than in the surrounding water. The degree of biomagnification was greater for the more highly chlorinated polychlorinated naphthalene mixtures. Biomagnification of polychlorinated naphthalenes also occurs in shrimp. Grass shrimp concentrate

TABLE 2

Water and Sediment Polychlorinated Naphthalene Levels

Industry	Type of Sample	Level (µg/kg or µg/l)	Reference
Airplane engine overhaul	Sediment	1250-5000	Crump-Wiesner, et al. 1973
Airplane engine overhaul	Water	5-7	Crump-Wiesner, et al. 1973
None identified	Sediment	55	Law and Goerlitz, 1974
Reprocessing oil	Sediment	trace	Minagawa, 1976
Polychlorinated Naphthalene manufacturer	Water	n.d[a].-1.4	Erickson, et al. 1978
Capacitor manufacturer A	Water	n.d.	Erickson, et al. 1978
Capacitor manufacturer B	Water	n.d.-0.6	Erickson, et al. 1978
Capacitor manufacturer B	Sediment	1.8-2.6	Erickson, et al. 1978
Capacitor dumps (2)	Water	n.d.	Erickson, et al. 1978

[a]n.d. means not detectable with a sensitivity threshold of 0.2 µg/l for water and 0.5 µg/kg for soil and sediment.

various mixtures of polychlorinated naphthalenes by a factor ranging from 63 to 257 compared to the surrounding water (Green and Neff, 1977). As with algae there is greater biomagnification with the more highly chlorinated naphthalenes. A bioconcentration factor (BCF) relates the concentration of a chemical in water to the concentration in aquatic organisms, but BCF's are not available for the edible portions of all four major groups of aquatic organisms consumed in the United States. Since data indicate that the BCF for lipid-soluble compounds is proportional to percent lipids, BCF's can be adjusted to edible portions using data on percent lipids and the amounts of various species consumed by Americans. A recent survey on fish and shellfish consumption in the United States (Cordle, et al. 1978) found that the per capita consumption is 18.7 g/day. From the data on the 19 major species identified in the survey and data on the fat content of the edible portion of these species (Sidwell, et al. 1974), the relative consumption of the four major groups and the weighted average percent lipids for each group can be calculated:

Group	Consumption (Percent)	Weighted Average Percent Lipids
Freshwater fishes	12	4.8
Saltwater fishes	61	2.3
Saltwater molluscs	9	1.2
Saltwater decapods	18	1.2

Using the percentages for consumption and lipids for each of these groups, the weighted average percent lipids is 2.3 for consumed fish and shellfish.

A measured bioconcentration factor of 2,300 was obtained for Halowax 1014 using brown shrimp containing about 1.1 percent lipids (U.S. EPA, 1976). Since this test only lasted 4 days and the result was based on whole body, this BCF is probably lower than the steady-state value. An adjustment factor of 2.3/1.1 = 2.1 can be used to adjust the measured BCF from the 1.1 percent lipids of the brown shrimp to the 2.3 percent lipids that is the weighted average for consumed fish and shellfish. Thus, the weighted average bioconcentration factor for Halowax 1014 and the edible portion of all aquatic organisms consumed by Americans is calculated to be 2,300 x 2.1 = 4,800.

Erickson, et al. (1978) also noted a higher level of polychlorinated naphthalenes in a dead fish (39 µg/kg) than in the adjacent water (0.2 µg/l).

Erickson, et al. (1978) also noted a higher relative biomagnification of the lowest chlorinated naphthalenes by the fruit of apple trees grown on contaminated soil. The soil was found to have a polychlorinated naphthalene level of 190 µg/kg of which 1.6 µg/kg consisted of monochloronaphthalenes. While the apples grown on this soil had only 90 µg/kg of polychlorinated naphthalenes, the level of monochloronaphthalenes was 62 µg/kg.

Inhalation

The American Conference of Governmental Industrial Hygienists (ACGIH) (1971), in the documentation of threshold limit values for polychlorinated naphthalenes, noted that in industry air concentrations of 1,000 to 2,000 $\mu g/m^3$ of a penta- and hexachloronaphthalene mixture and concentrations of 300 $\mu g/m^3$ of trichloronaphthalene (possibly with some tetrachloronaphthalene present) had been associated with adverse effects. Erickson, et al. (1978) found ambient air concentrations of polychlorinated naphthalenes ranging from 0.025 to 2.90 $\mu g/m^3$ near the Koppers' polychlorinated naphthalene plant. Concentrations of trichloronaphthalene were as high as 0.95 $\mu g/m^3$ while hexachloronaphthalene concentrations never exceeded 0.007 $\mu g/m^3$. Near one capacitor factory, ambient air concentrations of polychlorinated naphthalenes ranged from non-detectable to 0.005 $\mu g/m^3$, while at a second factory they ranged from 0.0098 to 0.033 $\mu g/m^3$.

Dermal

The likelihood of significant dermal absorption of polychlorinated naphthalenes from an environmental source is negligible. Link, et al. (1958) found no evidence of systemic disease after spraying pigs with 1,386 to 1,704 mg/kg of hexachloronaphthalene over a period of 28 days, while a total dose of only 41 mg/kg of hexachloronaphthalene given orally over a period of 10 days was uniformly fatal.

PHARMACOKINETICS

Absorption, Distribution, and Excretion

There is currently no information on the pharmacokinetic mechanisms of absorption, distribution and excretion of polychlorinated naphthalenes in man. Chu, et al. (1977a) noted that in rats fed 1,2-dichloronaphthalene, the half-life for loss of this compound from the blood after the first day was 24 hours. This chemical and its metabolites were found primarily in the intestine, kidney, and adipose tissue (Table 3). Although initially more of this chemical and its metabolites were found in the urine, a greater proportion had been excreted in the feces by the end of 7 days. A stool analysis disclosed only unchanged 1,2-dichloronaphthalene. In contrast, only a glucuronide-bound dihydrodiol metabolite of 1,2-dichloronaphthalene could be identified in the urine. Sixty-two percent of the dose was excreted in the bile in 24 hours compared to 18.9 percent lost in the feces in 24 hours. This suggests that there is an appreciable reabsorption and enterohepatic recirculation of this particular compound.

TABLE 3

Distribution and Excretion of 1,2-Dichloronaphthalene in Rats. (as a percentage of dose) (Chu, et al. 1977a)

	At 24 hours	At 48 hours	At 7 days
Adipose	0.1	0.15	0.04
Lung	0.04	0.03	---
Liver	0.7	0.07	---
Bladder	0.01	0.01	---
Intestine	0.45	3.6	---
Skin	0.07	0.08	0.01
Gastrointestinal content	18.3	17.9	0.04
Fecal excretion	18.9	30.8	42.0
Urine excretion	26.4	32.6	35.2

In seagulls with environmental exposures to chlorinated naphthalenes, analyses of fat, liver, and plumage resulted in the detection of polychlorinated naphthalenes only in liver samples, the highest value being 62,500 µg/kg calculated as octachloronaphthalene (Vannucchi, et al. 1978).

Metabolism

There appears to be appreciable metabolism in mammals of polychlorinated naphthalenes containing four chlorine atoms or less. Cornish and Block (1958) investigated the excretion of polychlorinated naphthalenes in rabbits. They found that 79 percent of 1-chloronaphthalene, 93 percent of dichloronaphthalene, and 45 percent of tetrachloronaphthalene were excreted in the urine as metabolites of the parent compounds. There was no measurable urinary excretion (either as metabolites or the unchanged compound) of penta-, hepta-, or octachloronaphthalene.

There have been detailed evaluations of the various urinary metabolites of polychlorinated naphthalenes as noted in Table 4. Thus, metabolism may involve hydroxylation alone or hydroxylation in combination with dechlorination.

Ruzo, et al. (1976) investigated the 1,2 shift of a chlorine atom during the metabolism of 1,4-dichloronaphthalene by substituting a deuterium atom for the one-position chlorine. The shift of the deuterium atom to the two-position suggested an arene oxide intermediary metabolite in the conversion of 1,4-dichloronaphthalene to 2,4-dichloro-1-naphthol. (See Figure 1).

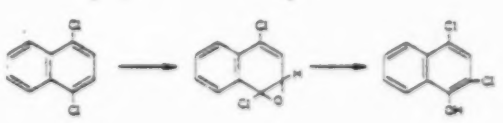

Figure 1. Conversion of 1,4-dichloronaphthalene to 2,4-dichloro-1-naphthol via an arene oxide intermediary metabolite. (Brinkman and Reymer, 1976).

TABLE 4

Polychlorinated Naphthalene Metabolites Found in Urine

Parent	Metabolite	Animal	Reference
1-chloronaphthalene	4-chloro-1-naphthol	frogs	Sundstrom, et al. 1975
		pigs	Ruzo, et al. 1976
2-chloronaphthalene	3-chloro-2-naphthol	pigs	Ruzo, et al. 1976
1,2-dichloro-naphthalene	3,4-dichloro-1-naphthol	pigs	Ruzo, et al. 1976
1,4-dichloro-naphthalene	2,4-dichloro-1-naphthol	pigs	Ruzo, et al. 1976
		frogs	Sundstrom, et al. 1975
2,6-dichloro-naphthalene	6-chloro-2-naphthol 2,6-dichloronaphthol	rats	Chu, et al. 1977b
2,7-dichloro-naphthalene	7-chloro-2-naphthol	rats	Chu, et al. 1977b
1,2-dichloro-naphthalene	5,6-dichloro-1,2-dihydroxy-1,2-dihydronaphthalene	rats	Chu, et al. 1977b
1,2,3,4-tetrachloro-naphthalene	5,6,7,8-tetrachloro-1- and -2-naphthol	pigs	Ruzo, et al. 1976
1,2,3,4,5,6-hexa-chloronaphthalene	none	pigs	Ruzo, et al. 1976

EFFECTS

In man the first disease that was recognized as being associated with exposure to polychlorinated naphthalenes was halowax acne (a form of chloracne) also known as "cable itch" or "cable rash." Occurrence of this disease was associated with the manufacture or use of polychlorinated naphthalene-treated electrical cables. During World War II chloracne was commonly found among shipyard electricians. Individuals who stripped the polychlorinated naphthalene-treated covering from cables would often contaminate their clothes with dust or flakes from the covering. If they wore their dirty work clothes home, their wives or children could get a milder form of chloracne (Schwartz, 1943). Chloracne has resulted both from skin contact and inhalation of polychlorinated naphthalene fumes. Polychlorinated naphthalenes dissolve readily and concentrate in the sebum material found in hair follicles (Jones, 1941). Initial symptoms are loss of the sebaceous glands emptying into the follicle, derangement of keratin formation, and plugging of the follicle with resultant comedo. If exposure stops at this point, the sebaceous glands can regenerate and the rash can clear after several months. Continued exposure injures the follicle walls causing an inflammatory reaction and formation of a pustule. Later, the walls deteriorate and rupture with loss of follicular material to the surrounding tissues. This results in the formation of a cyst or sterile abcess.

Not all polychlorinated naphthalenes are acneigenic. Shelley and Kligman (1975) applied various polychlorinated naphthalenes to human subjects. They found chloracne only after treating their subjects with a suspension containing a mixture of penta- and hexachlorinated naphthalenes. Similarly, Hambrick (1957) noted chloracne only after treating his subjects with a 3 percent solution of hexachloronaphthalene or a mixture of penta- and hexachlorinated naphthalenes. These were the only two mixtures that produced hyperkeratosis when applied to the ears of rabbits. Epidemiologic studies confirm these clinical and experimental impressions. Crow (1970) noted a continuing incidence of chloracne in a capacitor plant that utilized both tri-/tetrachlorinated and penta-/hexachlorinated naphthalene mixtures. As soon as the use of the latter mixture was stopped, chloracne ceased to be found at this factory. Kleinfeld, et al. (1972) noted that an electric coil manufacturing plant had no problems with chloracne while using a mono- and dichloronaphthalene mixture. When a tetra-/pentachlorinated naphthalene mixture was unwittingly substituted for the original mixture, 56 of the 59 potentially exposed workers developed chloracne within a short time. They also complained of puritis, eye irritation, headaches, fatigue, vertigo, nausea, loss of appetite, and weight loss. Liver function studies in five of the affected individuals were normal. Kimbrough and Chamblee (1972) give a general review of chloracne in industrially exposed populations.

Individuals with high-level exposures to the fumes of polychlorinated naphthalenes can develop acute or sub-acute liver disease with or without an associated chloracne. With a rapidly progressive course there is jaundice, abdominal pain, edema, ascites, and decrease in liver size. At autopsy the liver is small and necrotic with evidence of fatty metamorphosis, a condition called acute yellow atrophy. With less exposure the course can be long enough for the development of a postnecrotic-type of cirrhosis or liver scarring. At the time of death, common findings in addition to severe liver damage include evidence of damage to the heart, pancreas, gallbladder, lungs, adrenal glands, and kidney tubules (Greenberg, et al. 1939; Strauss, 1944). With even less exposure there may be few or no clinical findings and only mild-to-moderate laboratory evidence of liver dysfunction that resolves with time (Cotter, 1944).

Acute, Sub-acute, and Chronic Toxicity

Almost invariably, clinical evidence of damage from polychlorinated naphthalene exposure has occurred only after repeated exposures. Consequently, there have been few tests of acute toxicity. Cornish and Block (1957) in investigating metabolites of polychlorinated naphthalenes, gave groups of three rabbits single oral doses of various compounds at a level of 500 mg/kg and followed their course for 7 days. No mortality or illness occurred in the rabbits treated with mono-, di-, or tetrachloronaphthalenes. One of the three treated with pentachloronaphthalene died. All the rabbits treated with a solution of hepta- or octachloronaphthalene died.

Beginning in the 1930's a number of herds of cattle were afflicted with a mysterious 'X-disease' or hyperkeratosis of cattle. Severely afflicted individuals developed coarse, wrinkled skin, a chronic cough and shortness of breath, weight loss with associated inflammation of the upper portion of the gastrointestinal tract, pancreatitis and pancreatic scarring, kidney damage, gallbladder disease, severe liver damage, hair loss and reversible suppression of spermatogenesis (Vlahos, et al. 1955). In addition, animals were found to be more susceptible to a viral infection, proliferative stomatitis, which caused warty growths of the mucosal lining of the nose, mouth, and intestinal tract (Olson, 1969). This disease was eventually traced to the ingestion (either by licking farm equipment or by eating contaminated food pellets) of oil or grease containing polychlorinated naphthalenes. The investigation of the origins of this illness stimulated several studies on the subacute and chronic toxicity of polychlorinated naphthalenes taken orally. Although many of the studies were performed using calves or cattle (Table 5), a number of studies were done with several other species (Table 6). Polychlorinated naphthalenes containing three or fewer chlorine atoms were found to be nontoxic. Tetrachloronaphthalene resulted in mild liver disease at levels as high as 0.7 mg/kg/day (Bell, 1953). The higher chlorinated naphthalenes produced more severe disease at lower doses. Because of their insolubility, hepta- and octachloronaphthalene were less toxic when given in suspension than when given in solution.

TABLE 5

Oral Toxicity of Polychlorinated Naphthalenes in Cattle

No. of chlorine atoms	Dose (mg/kg/day)	Duration (days)	Results	Reference
2	0.9	7	no effect	Bell, 1953
3	0.49-0.54	7-10	no effect	Bell, 1953
4	0.48-0.70	10-18	slight hyperkeratosis	Bell, 1953
5	0.35-0.49	5-10	severe systemic disease	Bell, 1953
6	0.23-0.65	5-10	severe systemic disease	Bell, 1953
6	0.83-1.66	60	severe systemic disease	Sikes, et al. 1952
7	0.14-0.49	7-9	severe systemic disease	Bell, 1953
8	0.21-0.70	9-13	moderate systemic disease	Bell, 1953
8	0.88	13	severe systemic disease	Sikes, et al. 1952

TABLE 6

Oral Toxicity of Polychlorinated Naphthalenes

No. of chlorine atoms	Dose	Duration (days)	Results	Species	Reference
3,4	0.3 g/rat/day	136	slight liver damage	rats	Bennett, et al. 1938
4,5	50 mg/rat/day	63	all moribund or dead	rats	Bennett, et al. 1938
5,6	100 mg/rat/day	55	all moribund or dead	rats	Bennett, et al. 1938
5,6	0.23 mg/kg/day	135	cirrhosis	sheep	Brock, et al. 1957
5,6	2.3 mg/kg/day	23-35	all dead or moribund (acute yellow atrophy)	sheep	Brock, et al. 1957
6	2.3 mg/kg/day	10	no effect	pigs	Link, et al. 1958
6	3.4 mg/kg/day	8	slight decrease in Vitamin A	pigs	Link, et al. 1958
6	3.5 mg/kg/day		Marked decrease in Vitamin A	pigs	Link, et al. 1958
6	3.6 mg/kg/day	10	1 of 3 dead	pigs	Link, et al. 1958
6	4.1 mg/kg/day	10	3 of 3 dead	pigs	Link, et al. 1958

Synergism and/or Antagonism

Drinker, et al. (1937) exposed rats to an average of 1.31 mg/m^3 of trichloronaphthalene or to 1.16 mg/m^3 of a penta-/hexachloronaphthalene mixture in air for 6 weeks with only minor liver effects. When a similarly exposed group of rats was challenged with a sublethal dose of an ethanol/carbon tetrachloride mixture, no effect was seen with the trichloronaphthalene-exposed rats but 7 of the 10 penta-/hexachloronaphthalene-exposed rats died. No other data are available on potentially synergistic or antagonistic effects.

Teratogenicity, Mutagenicity, and Carcinogenicity

No animal or human studies have been carried out on the carcinogenicity, mutagenicity, or teratogenicity of polychlorinated naphthalenes.

CRITERION FORMULATION

Existing Guidelines and Standards

The only standards that presently exist for polychlorinated naphthalenes are the Occupational Safety and Health Administration's standards which were adopted from and are identical to the ACGIH Threshold Limit Values. The rigor of these standards increases as the number of chlorine atoms present increases on the assumption that vapor toxicity is proportional to the number of chlorine atoms present in each compound. The present Threshold Limit Values are:

Trichloronaphthalene	5 mg/m^3
Tetrachloronaphthalene	2 mg/m^3
Pentachloronaphthalene	0.5 mg/m^3
Hexachloronaphthalene	0.2 mg/m^3
Octachloronaphthalene	0.1 mg/m^3

There are no state or federal water quality or ambient air quality standards for chlorinated naphthalenes.

Current Levels of Exposure

Polychlorinated naphthalenes have not been identified in drinking water samples, market basket food samples, or at standard ambient air stations. Near point sources, concentrations in water can range as high as 7.0 µg/l (Crump-Wiesner, et al. 1973) and concentrations in air as high as 2.9 $\mu g/m^3$ (Erickson, et al. 1978). Near a point source one fish sample had a level of 39 µg/kg for the whole fish, and a sample of apples contained 90 µg/kg of polychlorinated naphthalenes (Erickson, et al. 1978). Polychlorinated naphthalenes have been detected in several samples of PCBs, compounds that

are known to be widely distributed in the aquatic environment. Measurements of chlorinated naphthalenes in environmental samples have not been widely performed using current sensitive measurement techniques for these compounds.

Special Groups at Risk

Because of the possible potentiation of the toxicity of higher chlorinated naphthalenes by ethanol and carbon tetrachloride, individuals who ingest enough alcohol to result in liver disfunction would be a special group at risk. Individuals (e.g., analytical and synthetic chemists, mechanics, and cleaners) who are routinely exposed to carbon tetrachloride or other hepatotoxic chemicals would also be at a greater risk than a population without such an exposure. Individuals involved in the manufacture, utilization, or disposal of polychlorinated naphthalenes would be expected to have higher levels of exposure than the general population.

Basis and Derivation of Criterion

There are insufficient animal toxicity data available on which to base a criterion for polychlorinated naphthalenes. However, industrial exposure to vapors of polychlorinated naphthalenes has resulted in systemic toxicity, and this toxicity is the basis for the present ACGIH threshold limit values (TLV). Such a TLV can be used as a basis for developing water criteria for polychlorinated naphthalenes. It is recognized that the ACGIH TLVs apply primarily to normal adult working males and do not incorporate safety factors for sensitive populations. In order to provide a reasonable margin of safety, calculation of an acceptable

concentration of polychlorinated naphthalenes in drinking water as proposed by Stokinger and Woodward (1958) should include a safety factor of 100 as illustrated below:

$$\frac{\text{TLV } (mg/m^3) \cdot 50\ m^3/wk}{7\ \text{days/wk} \cdot 100} = \text{acceptable intake (mg/day)}$$

where 50 m^3/wk = average amount of air inhaled by a normal adult in a 40 hour work week;

7 days/wk = conversion factor for daily intake;

100 = safety factor for sensitive populations (NAS, 1977).

Since no pharmacokinetic data are available to compare absorption efficiency by the inhalation route versus the oral route, it is assumed that absorption efficiency is the same by either route.

Using the ACGIH TLV levels, the acceptable daily intakes for polychlorinated naphthalenes would be as follows:

	Acceptable Daily Intake (mg)
Trichloronaphthalenes	0.36
Tetrachloronaphthalenes	0.14
Pentachloronaphthalenes	0.036
Hexachloronaphthalenes	0.014
Octachloronaphthalene	0.007

Assuming an average intake of 18.7 g of fish per day with a biomagnification factor of 4,800 for edible portions of aquatic species as derived in the Exposure section, and a water intake of 2 l/day, then criteria levels for the above polychlorinated naphthalenes in water would be as follows:

	Criterion Level (µg/l)
Trichloronaphthalenes	3.9
Tetrachloronaphthalenes	1.5
Pentachloronaphthalenes	0.39
Hexachloronaphthalenes	0.15
Octachloronaphthalene	0.08

REFERENCES

American Conference of Governmental Industrial Hygienists. 1971. Documentation of the Threshold Limit Values for substances in workroom air. Cincinnati, Ohio.

American Conference of Governmental Industrial Hygenists. 1977. TLVs Threshold Limit Values for chemical substances and physical agents in the workroom environment with intended changes. Cincinnati, Ohio.

Bennett, G.A., et al. 1938. Morphological changes in the livers of rats resulting from exposure to certain chlorinated hydrocarbons. Jour. Ind. Hyg. Toxicol. 20: 97.

Bell, W.S. 1953. The relative toxicity of the chlorinated naphthalenes in experimentally produced bovine hyperkeratosis (X-disease). Vet. Met. 48: 135.

Bowes, G.W., et al. 1975. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 256: 305.

Brock, W.E., et al. 1957. Chlorinated naphthalene intoxication in sheep. Am. Jour. Vet. Res. 18: 625.

Brinkman, U.A., and H.G.M. Reymer. 1976. Polychlorinated naphthalenes. Jour. Chromatog. 127: 203.

Chu, I., et al. 1977a. Metabolism and tissue distribution of (1,4,5,-^{14}C)-1,2-dichloronaphthalene in rats. Bull. Environ. Contam. Toxicol. 18: 177.

Chu, I., et al. 1977b. Metabolism of chloronaphthalenes. Jour. Agric. Food Chem. 25: 881.

Cordle, F. et al. 1978. Human exposure to polychlorinated biphenyls and polybrominated biphenyls. Environ. Health Perspect. 24: 157.

Cornish, H.H., and W.D. Block. 1958. Metabolism of chlorinated naphthalenes. Jour. Biol. Chem. 231: 583.

Cotter, L.H. 1944. Pentachlorinated naphthalenes in industry. Jour. Am. Med. Assoc. 125: 373.

Crow, K.D. 1970. Chloracne: a critical review including a comparison of two series of cases of acne from chlornaphthalene and pitch fumes. Trans. St. John's Hosp. Derm. Soc. 56: 79.

Crump-Wiesner, H.J., et al. 1973. A study of the distribution of polychlorinated biphenyls in the aquatic environment. Jour. Res. U.S. Geol. Survey 1: 603.

Drinker, C.K., et al. 1937. The problem of possible systemic effects from certain chlorinated hydrocarbons. Jour. Ind. Hyg. Toxicol. 19: 283.

Erickson, M.D., et al. 1978. Sampling and analysis for polychlorinated naphthalenes in the environment. Jour. Assoc. Off. Anal. Chem. 61: 1335.

Green, F.A., Jr., and J.M. Neff. 1977. Toxicity, accumulation, and release of three polychlorinated naphthalenes (Halowax 1000, 1013, and 1099) in postlarval and adult grass shrimp, Palaemonetes pugio. Bull. Environ. Contam. Toxicol. 14: 399.

Greenburg, L., et al. 1939. The systemic effects resulting from exposure to certain chlorinated hydrocarbons. Jour. Ind. Hyg. Toxicol. 21: 29.

Hambrick, G.W. 1957. The effects of substituted naphthalenes on the pilosebaceous apparatus of rabbit and man. Jour. Invest. Derm. 28: 89.

Jones, A.T. 1941. The etiology of acne with special reference to acne of occupational origin. Jour. Ind. Hyg. Toxicol. 23: 290.

Kimbrough, R.D., and M.D. Chamblee. 1972. Toxicity of chlorinated hydrocarbons and related compounds. Arch. Environ. Health 25: 125.

Kimbrough, R.D., et al. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1200. Jour. Nat. Cancer Inst. 55: 1453.

Kleinfeld, M., et al. 1972. Clinical effects of chlorinated naphthalene exposure. Jour. Occup. Med. 14: 377.

Law, L.M., and D.F. Goerlitz. 1974. Selected chlorinated hydrocarbons in bottom material from streams tributary to San Francisco Bay. Pestic. Monitor. Jour. 8: 33.

Link, R.P., et al. 1958. Toxic effect of chlorinated naphthalenes in pigs. Jour. Am. Vet. Med. Assoc. 133: 83.

Minagawa, K. 1976. Polychlorinated naphthalenes in the surrounding environment of a reproduction factory of used transformer oil. Jap. Jour. Ind. Health. 18: 416.

National Academy of Sciences. 1977. Drinking water and health. Washington, D.C.

Olson, C. 1969. Bovine hyperkeratosis. (X-diseases, highly chlorinated naphthalene poisoning). Historical review. Pages 101-157 in C.A. Bradly and C.E. Cornelius, eds. Advances in veterinary science and comparative medicine. Academic Press, New York.

Ruzo, L., et al. 1976. Metabolism of chlorinated naphthalenes. Jour. Agric. Food Chem. 24: 581.

Schwartz, L. 1943. An outbreak of halowax acne ("cable rash") among electricians. Jour. Am. Med. Assoc. 122: 158.

Shelley, W.B., and A.M. Kligman. 1957. The experimental production of acne by penta- and hexachloronaphthalenes. A.M.A. Arch. Derm. 75: 689.

Sidwell, V.D., et al. 1974. Composition of the edible portion of raw (fresh or frozen) crustaceans, finfish, and mollusks. I. Protein, fat, moisture, ash, carbohydrate, energy value, and cholesterol. Mar. Fish. Rev, 36: 21.

Sikes, D., et al. 1952. The experimental production of "X-disease" (hyperkeratosis) in cattle with chlorinated naphthalenes and petroleum products. Jour. Am. Vet. Med. Assoc. 121: 337.

Stokinger, H.E., and R.L. Woodward. 1958. Toxicologic methods for establishing drinking water standards. Jour. Am. Water Works Assoc. 50: 515.

Strauss, N. 1944. Hepatotoxic effects following occupational exposure to Halowax® (chlorinated hydrocarbons). Rev. of Gastroenterol. 11: 381.

Sundstrom, G., et al. 1975. Methods for the study of metabolism of toxic and persistent chemicals in aquatic organisms as exemplified by chloronaphthalenes. Pages 177-188 in Proc. Swedish Netherland Symp., Elsevier, Amsterdam.

U.S. EPA. 1976. Semi-annual report, Environ. Res. Lab., Gulf Breeze, Fla. April-Sept. U.S. Environ Prot. Agency.

Vannucchi, C., et al. 1978. Residues of chlorinated naphthalenes, other hydrocarbons and toxic metals (Hg, Pb, Cd) in tissues of Mediterranean seagulls. Chem-sphere 6: 483.

Vlahos, et al. 1955. Destruction and restoration of spermatogenesis in a bull experimentally poisoned with high chlorinated naphthalene. Cornell Vet. 45: 198.

Vos, J.G., et al. 1970. Identification and toxicological evaluation of chlorinated dibenzofurans and chlorinated naphthalenes in two commercial polychlorinated biphenyls. Food. Cosmet. Toxicol. 8: 625.

Walsh, G.E., et al. 1977. Effects and uptake of chlorinated naphthalenes in marine unicellular algae. Bull. Environ. Contam. Toxicol. 18: 297.